district. Pelvic inflammatory disease actually became a less common finding at laparotomy, though women using intrauterine contraceptive devices, who were a low risk group for a history of pelvic inflammatory disease and the finding of pelvic adhesions, had equivalent rates of endosalpingitis. Given that ectopic pregnancy has been linked with lower socioeconomic groups, it may be that the massive increase in unemployment has had more than a small part to play in the change in the rate of this disease over the past few years.

Supersonic jet lag

Dr GEOFFREY BENNETT (Civil Aviation Authority, London WC2B 6TE) writes: Dr Clifford Hawkins (21 March, p 766) finds from his reading that "supersonic travel will eliminate jet lag as the passengers' internal clock will not be disturbed." A keen passenger on Concorde, I wish it were so. Alas, it is not the travelling hopefully and speedily that affects my internal clock; it is the different time zone in which I arrive. And if I am travelling eastwards recent work shows that it may be much more than a week before all my circadian rhythms resynchronise.

75 Deaths in asthmatics prescribed home nebulisers

Drs DAVID J GODDEN, DAVID N K SYMON (University of Aberdeen, Aberdeen AB9 1FX), and DOUGLAS A ROBERTSON (Raigmore Hospital, Inverness IV2 3UJ) write: The conclusion of Dr P Ebden and coworkers (11 April, p 294) that 10 mg of salbutamol, delivered with an air driven nebuliser, has no clinically important effect on plasma potassium concentration requires qualification. Hypokalaemia has been observed after inhalation of β agonists by normal volunteers.[1,2] It has been suggested that asthmatics and non-asthmatics may differ in their hypokalaemic response to β agonist treatment,[3] but whether this represents a true intrinsic difference or acquisition of tolerance after repeated dosing remains unclear.[4]

We compared the effects of serum potassium concentration of inhaled nebulised salbutamol (5 mg) with those of placebo in 10 stable subjects with asthma, mean age 32 years. The mean (SD) baseline serum potassium concentration on the placebo day was 4·0 (0·3) mmol/l and on the salbutamol day 4·0 (0·2) mmol/l. Serum potassium concentration was estimated 30 and 60 minutes after salbutamol and placebo were given, and concentrations remained stable on both days. The mean serum potassium concentration after salbutamol was 4·0 (0·3) mmol/l and after placebo 4·2 (0·2) mmol/l. After receiving salbutamol, the three subjects who had received no previous β agonist treatment showed a fall in serum potassium concentrations of 0·3, 0·3, and 0·6 mmol/l, respectively. This raises the question whether regular dosing with β agonists protects against hypokalaemia as a result of tolerance and, conversely, whether subjects with asthma receiving no regular treatment with β agonists may indeed develop clinically significant falls in serum potassium concentration after therapeutic doses of inhaled nebulised β agonist. While we agree, therefore, that the standard therapeutic doses of inhaled salbutamol do not seem to cause clinically important hypokalaemia in patients with asthma who have undergone previous treatment, we believe that the effects on subjects who have received no previous treatment require further examination.

1 Smith SR, Ryder C, Kendall MJ, Holder R. Cardiovascular and biochemical responses to nebulised salbutamol in normal subjects. *Br J Clin Pharmacol* 1984;18:641-4.
2 Haalboom JRE, Deenstra M, Struyvenberg A. Hypokalaemia induced by inhalation of fenoterol. *Lancet* 1985;i:1125-7.
3 Monie RDH, Hartley JPR, Winson MD. Metabolic responses to inhaled salbutamol. *Br J Dis Chest* 1980;74:316.
4 Smith SR, Kendall MJ. Metabolic responses to beta-2-stimulants. *J R Coll Physicians Lond* 1984;18:190-4.

Dr DESMOND MURPHY (St Mary's Hospital, Newport, Isle of Wight PO30 5TG) writes: I was surprised by the absence of "failure of power supply" as a factor in any of the 75 deaths in patients with asthma who had home nebulisers in New Zealand, as described by Dr M R Sears and colleagues (21 February, p 477). I have also been surprised by the absence of any subsequent letters on the subject from Britain. In the study by Dr Sears and coworkers all the patients were probably using nebulised β agonists for episodes of acute severe asthma. In 1983 on the Isle of Wight a young woman died of acute severe asthma after her home nebuliser became unusable because of a power failure, owing to storm damage to electricity lines. The coroner refused to treat this as death from natural causes, accepting that she would have survived if the treatment had been available. He concluded that the death was accidental. In my recently completed survey of 153 patients currently using home nebulisers 28 had had their treatments affected by technical or power failure, and it was obvious from their comments that great anxiety ensued. If, as seems likely, home nebuliser treatment for acute severe asthma becomes more popular in Britain more lives may be lost unless patients have a secondary source of electrical supply or a foot pump nebuliser or some form of manually operated nebuliser. This requirement should further limit the number of patients in whom β agonist treatment by nebuliser is appropriate for the emergency treatment of asthma in the home.

Autologous blood transfusion

Mr A HEDLEY BROWN (Freeman Hospital, Newcastle upon Tyne NE7 7DN) writes: In response to Mr Alberic G T W Fiennes's comments (7 March, p 648), we believe that underreplacement of perioperative blood losses may be advantageous in coronary artery surgery. Before aspirin and dipyridamole were used we showed that those leaving hospital with haemoglobin concentrations below 115 g/l had better long term freedom from angina than those whose haemoglobin concentrations at discharge were higher. Our suspicion was that this was because there was a higher coronary flow rate and therefore less chance of thrombogenically slow flows through the grafts in these fairly anaemic subjects. We have shown that in dogs pure haemodilution of coronary blood without simultaneous dilution of the systemic blood causes an increased coronary flow rate in normal animals. Of course, in patients the increased cardiac output of anaemia would correspondingly increase myocardial demands on the coronary circulation, increasing the flow still further. It seems that the disadvantages of transfusing above haemoglobin concentrations of 115 g/l outweigh the advantages and that the use of blood as volume replacement should be minimised.

Deep vein thrombosis after stroke

Dr P A O'NEILL (Department of Geriatric Medicine, University Hospital of South Manchester) writes: In reply to a recent question (28 February, p 563) C W Havard stated that a deep vein thrombosis was unlikely to develop during the first week after a stroke. This is misleading as it has been clearly shown by Warlow and colleagues,[1] and confirmed by McCarthy and Turner,[2] using iodine-125 fibrinogen scanning, that thrombi begin to form within a few days after a stroke. These studies indicate that the mechanisms leading to major deep vein thromboses and death due to pulmonary emboli are triggered soon after the stroke has occurred. As in other circumstances in which deep vein thrombosis are common research should therefore be directed towards preventing the formation of thrombi from the outset either by treatment with subcutaneous heparin[2] or by physical methods.

1 Warlow C, Ogstan D, Douglas AS. Deep venous thrombosis of the legs after strokes. *Br Med J* 1972;i:1178-83.
2 McCarthy ST, Turner J. Low dose subcutaneous heparin in the prevention of deep vein thrombosis and pulmonary emboli following acute stroke. *Age Ageing* 1986;15:84-8.

Erythema migrans borreliosis or Lyme disease?

Dr JAMES HAWORTH (CH-1292 Chambésy, Switzerland) writes: Dr Ann Parke's leading article is a masterpiece of conciseness on the clinical features of a form of erythema migrans borreliosis as it occurs in New England under the name of Lyme disease (28 February, p 525). It does less than justice, however, to the history and epidemiology of the condition that until recently was called erythema chronicum migrans and was found by Arvid Afzelius in Sweden in 1909 and chronicled since then in Scandinavia, Germany, Austria, France, and Switzerland. Its association with the tick was equally documented by Afzelius (1910) and by Lipschültz (1913). The disease is being diagnosed with increasing frequency in France, the Federal Republic of Germany, and Switzerland (as well as Britain) and will undoubtedly spread geographically and in intensity over the summer, endangering nature lovers. At the Ninth International Congress of Parasitology of Infectious and Parasitic Diseases in 1980 the name of erythema migrans borreliosis was generally accepted, though Lyme borreliosis was also used. It seems to be a retrograde step to rename the entire entity using a recently discovered focus in Lyme, USA, when the use of "erythema migrans borreliosis" would keep the historical dermatological association of the disease along with its causative organism, thereby drawing attention to the possible complications.

Preventing AIDS

Dr ALEX COMFORT (Cranbrook, Kent TN17 3AH) writes: I am probably not alone in my concern over the party line adopted by the government over the prevention of the acquired immune deficiency syndrome (AIDS), which focuses entirely on the condom. The logic is sound: condoms do protect if properly used, but experience suggests that only those least at risk will use them. More serious is the fact that only very confident women may feel able to insist on them; young girls have enough difficulty in handling the importunate male and insisting on contraception, though here they have the contraceptive pill to their aid.

It is critically important that women have a means of self defence which does not depend on male cooperation. The obvious recourse would be the existing contraceptive foams based on nonoxynol-9, or improvements thereon. One is loth to advise them as a total substitute for barrier methods in view of the lack of a proper trial. There is, however, a classical instance in history where genital antiseptics proved superior both to condoms and to exhortation—the reduction of sexually transmitted diseases in troops on leave during the first world war. The antiseptics were crude (mercury ointment), but they worked. The continuance of this trial among civilians at risk was prevented, once the war was over, by an organised uproar about "condoning immorality." Had it been pursued, we might have controlled the sexually transmitted diseases then current long before we cured them. Developing an effective, harmless, and universally acceptable genital antiseptic would be cheaper, quicker, and more lifesaving in any explosive phase of spread than vaccines now in development. It will be needed long after AIDS is curable, to deal with human papillomavirus, herpes, trichomoniasis, and the other second rank sexually transmitted diseases, as well as any new infections now localised which may spread, as they will, in the "global village."

Portraits from memory

Major General M H P SAYERS (Walmer, Kent CT14 7LP) writes: Sir James Howie's fascinating tribute to the late Lieutenant Colonel Harold Bensted (11 April, p 959) contains an error in the penultimate paragraph, which I am sure he will forgive me for correcting. We owe it to those who struggled so hard at the sharp end of the scrub typhus scene in Burma during the second world war to point out that the scrub typhus vaccine was certainly tested in the field in a controlled trial in which some 10 000 men were vaccinated. The results were published in 1947[1] and for the reasons explained were inconclusive.

1 Card WI, Walker JM. Scrub typhus vaccine field trial in South-East Asia. *Lancet* 1947;i:481-3.

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